

Trichinosis in Saskatchewan An outbreak due to infected bear meat

H. E. Emson M.A., M.D., M. A. Baltzan, M.D., H. E. Wiens, M.D.,
Saskatoon, Sask.

While the incidence of reported trichinosis in the more populated regions of Canada has declined with improvement in feeding and inspection of hogs, the vector population of wild carnivores remains unchanged in the Canadian North and despite the influence of changes in human eating habits, the disease remains endemic there. Relatively few cases are diagnosed, however, and the present report concerns an outbreak of trichinosis in northern Saskatchewan from the consumption of undercooked bear meat, presumably from a black bear (*Ursus americanus*).

Case history

In early November, 1970 a small band of Chippewyan Indians from Wollaston Lake, Saskatchewan, was on a trapping expedition when they found and shot a hibernating bear for food. The bear meat was boiled for approximately one hour and then eaten, and about 10 days after its consumption the seven members of the hunting party, six men and one woman, became ill. They developed fever, chills and pains in the

muscles, joints and extremities, together with puffiness and swelling of the legs, hands and periorbital regions. Associated with these symptoms were generalized weakness, fatigue and dyspnea, but there was no change in bowel habit and no nausea or vomiting. The history was difficult to obtain as the patients spoke little but Chippewyan. Because of the symptoms all seven members of the hunting party were transported to hospital in Lac La Ronge, Saskatchewan, where examination confirmed the findings mentioned and laboratory tests showed leukocytosis with marked eosinophilia. A clinical diagnosis of trichinosis was made and two patients were transferred to St. Paul's Hospital, Saskatoon for confirmation of the diagnosis and possible therapy.

Case 1

Alphonse D., aged 25, was an acutely ill young Indian male. On admission his temperature was 103°F., pulse 110, respirations 30 and blood pressure 135/80. Periorbital edema was present and there was shotty cervical adenopathy. The liver was slightly enlarged. The arms and legs showed diffuse non-pitting edema

and were extremely tender. There were no other physical findings.

Results of laboratory investigations were as follows: Urinalysis was negative. Hematology: hemoglobin 15 g. per 100 ml., leukocyte count 13,200 per c.mm.; differential count, neutrophils 41%, bands 9%, eosinophils 40%, basophils 1% and lymphocytes 9%; platelets 701,000 per c.mm., ESR 9 mm. in the first hour. Serum creatinine 1.2 mg., urea 28 mg. per 100 ml., SGOT 116 units, LDH 1556 units per 100 ml.

Chest x-ray showed some fibrotic changes at the upper margin of the left hilus with knuckling of the aortic arch. Tomograms suggested enlarged lymph nodes in the mediastinum and aortic region.

A biopsy from the right forearm confirmed the presence of larvae of *Trichinella spiralis* in the skeletal muscle (Fig. 1).

Treatment with nematocidal agents was considered but was felt to be contraindicated in view of their marked toxicity and dubious clinical benefit.¹ After four days in hospital during which no medication was given, prednisone 15 mg., four times daily, was started. The temperature returned to normal within 36 hours and remained within normal limits thereafter. There was marked amelioration of all symptoms with decrease in pain, tenderness and swelling. The prednisone dosage was tapered rapidly in view of the possibility of occult tuberculosis; the patient was discharged on a dose of 30 mg. daily and returned to Lac La Ronge.

He was seen at intervals during the following year and appeared to have made a complete recovery with no residual symptomatology.

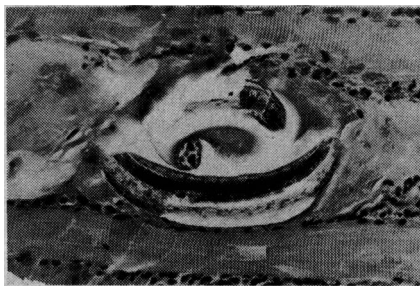


FIG. 1—*Trichinella spiralis* larva in skeletal muscle. Hematoxylin and eosin x 400.

H. E. EMSON, M.A., M.D., Director of
Laboratories, St. Paul's Hospital,
Saskatoon, Sask.

M. A. BALTZAN, M.D., Chief of Internal
Medicine, St. Paul's Hospital, Saskatoon,
Sask.

H. E. WEINS, M.D., General Practitioner, Lac
La Ronge, Sask.

Reprint requests to Dr. H. E. Emson,
Director of Laboratories, St. Paul's Hospital,
1702-20th Street West, Saskatoon,
Saskatchewan. S7M 029.

Case 2

Anne D. was an Indian girl of 16 years, the wife of the first patient described. The history and physical findings were similar to those of her husband. She was approximately five months pregnant and had apparently received no antepartum care. Urinalysis showed many leukocytes and culture of a mid-stream urine specimen produced a heavy growth of *Enterococcus*. Her ESR was 40 mm. and chest x-ray was normal. A biopsy of the gastrocnemius muscle confirmed the presence of larvae of *Trichinella spiralis*.

She was started on prednisone, 40 mg. per day, on the sixth hospital day, and discharged on a dosage of 7.5 mg. four times daily.

On March 19, 1971 she delivered a full-term normal male infant weighing 8½ lbs., which appeared perfectly healthy on careful examination. A year after her original infestation the mother appeared in good health, but a persistent leukocytosis of 11,000 per c.mm. was present, with 7% eosinophils. The cause of this is not known; no investigations for parasites were done at this time.

Discussion

The last reported outbreaks of trichinosis in Canada were due to pork.^{1, 2} In one outbreak thiabendazole was used in the treatment of 11 cases but the author concluded that it conferred no definite benefit.¹

The black bear has been recognized as a vector of trichinosis in the Arctic and cases in Alaska, Labrador and Northern Russia are reported.³⁻⁵ We are not aware of a previous report from the Prairie provinces.

The transplacental transmission of trichinosis with intrauterine infestation of the fetus has been reported in experimental animals, and in presumptive cases in humans.^{6, 7} The paucity of reports suggests that this must be a rare occurrence; despite the presumably heavy infection in Case 2, no abnormality was found in the child at delivery.

Résumé

Epidémie de trichinose en Saskatchewan, causée par consommation de viande d'ours avariée

Les auteurs rapportent une épidémie de trichinose dans une tribu d'Indiens Chippewayens vivant dans le Nord de la Saskatchewan et qui avaient mangé de la viande d'ours insuffisamment cuite. Le diagnostic a été basé sur l'anamnèse et sur l'examen clinique et confirmé par biopsie musculaire et par la découverte d'une forte éosinophilie. Les

sept malades ont guéri. Les deux malades qui ont été étudiés de façon plus approfondie ont été traités uniquement par la prednisone et ont guéri sans incidents: un an plus tard, on ne notait aucune séquelle. Une femme qui était enceinte de cinq mois au moment de l'infection a donné le jour à un enfant à terme et parfaitement normal.

References

1. THIBADEAU Y, GAGNON JJ: Trichinosis — Thiabendazole in the treatment of 11 cases. *Can Med Assoc J* 101: 533-535, 1969

2. BARR R: Human trichinosis. *Can Med Assoc J* 95: 912-917, 1966
3. RAUSCH RL, in Gould, S.E. (ed): *Trichinosis in Man and Animals*. Springfield, Illinois, USA; Charles C Thomas, 1970: p 368
4. MAYNARD JE, PAULS FP: Trichinosis in Alaska. *Am J Hyg* 76: 252-261, 1962
5. COFFEY JE, WIGLESWORTH FW: Trichinosis in Canadian Eskimos. *Can Med Assoc J* 75: 295-299, 1956
6. KUITUNEN-EKBAUM E: The incidence of trichinosis in humans in Toronto. *Can J Public Health* 32: 569-573, 1941
7. BOURNS TKR: The discovery of trichina cysts in the diaphragm of a 6 week old child. *J Parasitol* 38: 367, 1952

Trichinosis presenting as acute myocardial infarction

Gordon J. Kirschberg, M.D., *New York, N.Y.*

Summary: A 20-year-old male patient is presented as a case of trichinous myocarditis with clinical symptoms and electrocardiographic evidence of an acute inferior myocardial infarction. He recovered rapidly and completely without any specific therapy. This seems to be a distinct rarity, having never been previously reported, but is of importance because of the almost uniformly excellent prognosis in this condition in contradistinction to that of a bona fide myocardial infarction occurring at this age.

Although in 1936 Cushing¹ stated that "cases of trichinosis with clinical and ECG evidence of myocardial damage are rare", more recent reviews have shown that among patients with clinically apparent trichinosis, myocardial involvement as witnessed by ECG abnormalities occurs in between 21 and 23%.^{2, 3} These abnormalities include conduction defects, non-specific T-wave flattening or inversion, and arrhythmias.^{1, 2, 4-6} However, there has never been reported a case of trichinous myocarditis presenting

with focal ischemic changes resembling an acute myocardial infarction. Such a case was seen at the Royal Victoria Hospital in July 1968.

Case report

A 20-year-old Sicilian-born male restaurant kitchen helper was admitted because of severe retrosternal pain of several hours' duration. Family history was entirely negative and the patient had never been ill prior to this episode. He had not left Canada since his arrival two years previously.

Two days before admission the patient noted low grade fever, malaise, and vague soreness in his shoulders and thighs. On the day of admission he experienced three episodes of severe dull retrosternal pain without radiation, each lasting 10 minutes, and all occurring within a few hours. He did not vomit, nor did he experience dyspnea, orthopnea, or other discomfort. He had not had a sore throat, joint pains, urinary discoloration or other symptoms.

Physical examination revealed a healthy looking young man in moderate distress due to chest pain as described above. Oral temperature was 100° F., pulse was 88 and regular, blood pressure was 120/80. He had 1+ periorbital edema but no edema elsewhere. Findings on examination of the chest were normal. Auscultation of the heart revealed an atrial gallop without other abnormalities. There was slight deltoid tenderness but no neurological or any other abnormality.

An electrocardiogram done in the emergency room showed insignificant

From the Joint Cardiorespiratory Division, Royal Victoria Hospital, Montreal
Reprint requests to: Dr. Gordon J. Kirschberg, Neurological Institute, 710 W. 168th Street, N.Y., N.Y. 10032